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Oliguria in Surgical Patients

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• **The correction of the various causes of diminished urinary flow is of utmost importance in the preparation of patients with acute surgical conditions for operation. It has been demonstrated that adequate evaluation and correction of these factors are effective in reducing the high mortality accompanying severe trauma, late intestinal obstruction, rupture of an abdominal viscus and other surgical emergencies. The proper use of whole blood, plasma and saline is essential in the correction of hypovolemic states encountered in these conditions. This must be accomplished in most instances before surgical correction of the underlying disease is undertaken. Urinary flow is a valuable guide as to the effectiveness of replacement therapy.**

Oliguria after operation may result from a continuation of the factors causing the diminution of urinary flow before operation. The treatment used in the correction of the hypovolemia, as well as the surgical procedure, may contribute additional factors productive of a diminished urinary flow in the postoperative period.

A SEVERE REDUCTION in the formation of urine does not necessarily indicate a pathological state, but the early detection of oliguria is essential to the proper management of a seriously ill patient. Oliguria is an extremely valuable index of compensatory vasoconstriction secondary to the acute hypovolemia that characterizes surgical shock.

Oliguria is not a rigid term, for normally there is a wide variation in urine output. Anuria (zero output) is a rarity and usually indicates a mechanical obstruction to urine outflow or a massive vascular lesion, such as bilateral renal infarction or aortic occlusion. Under normal conditions, approximately 1 ml. of urine leaves the collecting ducts of the kidneys per minute; however, in the presence of very low water intake a urinary output of 500 ml. in 24 hours is considered volumetrically adequate (although this is, relatively, oliguria), if the concentration is high. A urine output of less than 400 ml. in 24 hours (about 17 ml. per hour) is definite oliguria.[11] A surgical patient seen initially with a urine output less than 17 ml. per hour must be considered definitely oliguric on the basis of hypovolemia and vasoconstriction. Equally indicative of acute hypovolemia is the sudden fall in hourly urine output in a normotensive postoperative patient whose output is being monitored with an indwelling catheter. This is usually the first clear objective indication of the situation.

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In an analysis of the causes of an acutely diminished output of urine, the primary consideration is recognition of factors that produce ischemia in a kidney. If these factors are present and are not reversed, the prolonged ischemia and anoxia may produce acute renal failure, especially if there is associated sepsis. Oliguria, as a result of oligemic hypotensive renal ischemia, coexistent with severe systemic infection, usually denotes such impending renal failure. Renal resuscitation instituted upon early detection of the oliguria will effectively reduce morbidity and mortality. Such resuscitation is de-

pendent upon correction of the decreased blood volume.

Reduction in glomerular pressure and rate of glomerular filtration with decreased urine formation is the primary effect of a diminished renal blood flow. The causative hypovolemia may be accompanied by a normal and, later, a lowered arterial systolic pressure. Although the peripheral systolic arterial pressure may be at normal levels because of generalized compensatory vasoconstriction, hypovolemia has a selective precocious vasoconstrictive effect on the kidney, with resulting early renal ischemia. Frequently in this situation the existence of normotensive shock is indicated by the presence of oliguria.

In addition to oliguria, other clinical features aid in the recognition of compensated shock.[2] In this latter situation when the patient with a normal systolic pressure in the supine position is then placed in a sitting position, there is usually a sharp reduction in the brachial blood pressure, often accompanied by vertigo or syncope. The radial pulse volume is decreased and peripheral veins are contracted. The feet are cool and pale. Oliguria associated with such findings heralds arterial hypotension.[8] Therefore, volumetric measurements of urine output usually reveal acute hypovolemia earlier than the sphygomanometer. The usual clinical stigmata of "shock," such as arterial hypotension, tachycardia and cold sweaty skin, are findings characteristic of decompensated shock.

Correction of reduction in effective circulating blood volume takes precedence in the management of a hypovolemic patient. Operative procedures should not be undertaken until the circulating blood volume has been restored to near normal, except in event of uncontrollable hemorrhage. The restoration of good urine output is an excellent parameter of the adequacy of repletion.

POOLED EXTRACELLULAR FLUID

It is customary to refer to the fluids and electrolytes in the body as existing in two major compartments, the intracellular and the extracellular. The intracellular portion, with potassium the dominant cation, constitutes approximately two-thirds of the body water. The remaining one-third of the body water, predominantly a sodium solution, is extracellular, and is largely located interstitially and in the vascular compartment. The extracellular fluid compartment also includes fluids contained in special systems and "potential" body spaces—gastrointestinal, cerebrospinal, ocular, peritoneal, pleural, pericardial and synovial. The fluid in these latter areas is "transcellular"[6] and is of slightly different composition than the interstitial fluid and plasma because of restricted diffusion and the active secretion of the cells lining these areas. This normal space might very properly be called the "third space." In current clinical usage, *extracellular* is used to describe the combined vascular and interstitial fluids. The rapid loss of important quantities of these fluids produces acute hypovolemia.

External or visible losses of extracellular fluid due to hemorrhage, vomiting, diarrhea or excessive perspiration are easily recognized. In physical, chemical, thermal and bacterial injuries, acute shifting of extracellular fluid into the region of the injury occurs. Such losses, due to body fluids that are shed internally, are not readily recognized. The overt swelling seen in burns or of an edematous limb in acute phlebitis readily reveals that such acute shifting has occurred. Frequently, losses are totally invisible, as when the internally trapped fluid accumulates as a pool in a body cavity or in tissue spaces.[9] The terms *third space, third compartment, obligatory sequestration* and others have been applied to this acutely pooled extracellular fluid. These terms have been of inestimable value in stressing this important concept. However, in some instances the terms have caused confusion in implying that a new anatomical space has been created. Also, the intravascular fluid is frequently designated as third compartment fluid. We prefer the term *pooled extracellular fluid.*

The majority of seriously ill patients with acute lesions requiring surgical operation have an occult loss of extracellular fluid. An example of this is the preperitoneal and intraperitoneal accumulation of dilute plasma secondary to diffuse chemical peritonitis resulting from perforated peptic ulcer. Similar sequestration of fluid also occurs in the tissues around an acutely inflamed pancreas. The trauma of major abdominal operation creates the same effect. In such conditions there is an acute and rapid loss of vascular fluid into the area of injury. This fluid loss results in the compensatory movement of extracellular fluid from noninjured areas into the vascular compartment. Whole blood, dilute plasma, and transcellular fluid such as intestinal contents (water, sodium, potassium) may comprise this pool of fluid, which is static and as unavailable to the organism as if it had been externally shed. Such acute pooling, unless counteracted by restoration therapy, causes a reduction in the amount of circulating blood volume and contraction of the active extracellular fluid compartment. Under such conditions, where a sizable static extracellular fluid pool develops, and whole blood is not lost, hemoconcentration inherently follows. Therefore, in hypovolemia of this type, the hematocrit is a direct measurement of the degree of hypovolemia. If plasma is used for correction, the hematocrit is also

an excellent guide to the volumetric adequacy of restoration of fluid. Unless there is renal damage, the urine output is an index of the homeostatic and physiological restoration of blood volume. Upon eradication of the mechanism that brought about the pooling, reversal occurs, and the pooled fluid reenters the vascular system. This will be indicated by a consequent increase in urine output.

RESTORATION OF CIRCULATING BLOOD VOLUME

In a hypovolemic or potentially hypovolemic surgical patient, the use of an indwelling urethral catheter to measure the hourly urine output is essential to good care. Also, repeated hematocrit or hemoglobin determinations must be obtained; and it is highly desirable to monitor the venous pressure, which can be done by means of a polyethylene catheter that has been inserted into the subclavian vein via a "cut-down" into a cephalic or antecubital vein. This polyethylene tube is also used for the administration of replacement fluids. Elevated venous pressure, especially in a geriatric patient, may indicate the need of digitalization for probable cardiac failure. The presence of such occult failure may be verified by a positive hepatojugular reflex and a prolonged circulation time.

The clinical evaluation of the patient's disease will indicate in most instances the type of fluid lost and reveal what is necessary for proper restoration (see Table 1). With normal kidneys, and in the absence of diabetes mellitus, an increase of urine flow to 30 ml. or more per hour following replacement usually indicates an important improvement in the circulating blood volume and reversal of sympathetic and hormonal action on the kidney. Hypovolemic hypotension should be treated by blood volume restoration rather than by vasoconstrictors. Such pressor drugs produce renal ischemia and reduced renal blood flow.[11]

When analysis of the disease mechanisms has been made and the type of fluid loss deduced, replacement may be planned. The fluid deficit may be merely water owing to inadequate intake and may be corrected by the use of dextrose in water, given slowly. Water, sodium and potassium deficits are primarily the result of excess loss of fluid from the gastrointestinal tract. The gastric fluid that is shed as a result of pyloric obstruction caused by duodenal ulcer reduces the blood volume and produces metabolic alkalosis. Initial replacement with dextrose and normal saline solution should be instituted and potassium added as soon as adequate renal function has been demonstrated. Enteric fluid losses due to intestinal obstruction, fistulas or diarrhea are frequently accompanied by metabolic acidosis; the initial repletion fluid of choice is M/6

TABLE 1.—*Differential Diagnosis of Oliguria*

I. Due to diminished renal blood flow ("prerenal")
- A. Water deficit
  - 1. Diminished oral intake
  - 2. Insufficient parenteral fluids
- B. Water and salt loss
  - 1. Vomiting
  - 2. Gastric suction
  - 3. Bowel obstruction
  - 4. Diarrhea
    - a. Bacterial
    - b. Pseudomembranous enterocolitis
    - c. Ulcerative colitis
  - 5. Gastro-intestinal fistulas
    - a. Pancreatic
    - b. Biliary
    - c. Duodenal
    - d. Small bowel
    - e. Gastrojejunocolic
    - f. Ileostomy malfunction
- C. Water, salt, and protein loss (plasma loss)
  - 1. Surface or external
    - a. Burns
  - 2. Internal
    - a. Traumatic operative edema and effusion
    - b. Peritonitis ("peritoneal burn")
      - (1) Enterogenous
        - (a) Ruptured peptic ulcer
        - (b) Perforative appendicitis
        - (c) Other intestinal perforations
    - c. Pancreatitis
    - d. Intestinal obstruction
    - e. Acute thrombophlebitis
    - f. Acute severe sepsis
    - g. Hypersensitivity reactions (anaphylaxis)
- D. Whole blood losses
- E. Mixed losses
  - 1. Severe wounds and trauma
- F. Other causes of renal ischemia
  - 1. Deep general anesthesia
  - 2. Vasoconstrictor drugs

II. Due to renal lesions ("renal")
- A. Acute renal failure ("tubular necrosis") resulting from
  - 1. Prolonged renal ischemia and anoxia
  - 2. Renal ischemia complicated by
    - a. Sepsis
    - b. Free pigments
      - (1) Transfusion reactions (hemoglobin)
      - (2) Crushed muscle (hemoglobin and myoglobin)
      - (3) Hepatic insufficiency (bile pigments)
    - c. Chemical toxins
      - (1) Sulfonamides
      - (2) Heavy metal ingestion
    - d. Preexisting renal disease
- B. Primary nephropathies
  - 1. Glomerulonephritis
  - 2. Other parenchymal lesions (rarely such diseases as pyelonephritis, collagen disease and others)
- C. Massive vascular lesions
  - 1. Bilateral renal infarction
  - 2. Aortic occlusion

III. Due to mechanical obstruction to urine flow
- A. Blocked catheter
- B. Urethral obstruction
  - 1. Bladder neck obstruction
  - 2. Strictures
- C. Ureteral obstruction
  - 1. Accidental ligation
  - 2. Calculi
  - 3. Tumors

sodium lactate. Saline solution and potassium are added when the oliguria has been corrected. Virus-free plasma (room-stored according to the method of Allen[1]) is now commercially available and its use is strongly recommended to rapidly correct severe acute hemoconcentration. It specifically restores plasma volume lowered from any cause. In the absence of anemia, plasma is preferable to whole blood because it eliminates the danger of viral hepatitis[4] and reactions, both of which may follow whole blood transfusion.

Plasma is the specific replacement fluid in those conditions in which water, salt, and albumin are lost from the effective circulating plasma. The peritoneal burn in chemical and bacterial peritonitis is accompanied by such losses due to acute pooling. In addition to plasma, M/6 sodium lactate is simultaneously administered volume for volume. As acidosis frequently accompanies these conditions, chloride solutions are avoided, especially if the danger of acute renal failure is anticipated. In the presence of good renal function, Ringer's lactate solution is used.

When plasma restoration is considered necessary for treatment of hemoconcentration, the plasma deficit should be estimated. This is accomplished by using the observed hematocrit and the following formula as suggested by Moore:[7]

$$\text{Blood volume} \times \text{Hematocrit} = \text{Red cell volume}$$

$$\text{Plasma deficit} = BV_1 - \frac{BV_1 \times Hct_1}{Hct}$$

Where BV_1 = Estimated normal blood volume
Hct_1 = Estimated normal hematocrit
Hct = Measured hematocrit

Except for the observed hematocrit, the remainder of the formula requires estimates of normal levels. "Normal" blood volume is estimated to be 7 per cent of the body weight, in nonobese patients, and 42 per cent is used as an average figure for the normal hematocrit.

A 70 Kg. lean male with a perforated duodenal ulcer and a hematocrit of 57 per cent would have the plasma loss estimated as follows:

$$BV_1 = (.07 \times 70{,}000) = 4900$$

$$\text{Plasma deficit} = 4900 - \frac{(4900 \times .42)}{.57}$$

$$\text{Deficit} = 1300 \text{ ml.}$$

Although it is a gross method of estimation, the formula is extremely helpful in planning plasma therapy. It frequently reveals the need for more replacement fluid than would otherwise have been given. However, whether or not this formula is used, repeated hematocrit or hemoglobin determinations following replacement are excellent guides.

In the presence of hemorrhage, dextran or plasma may be used for initial replacement while awaiting properly typed and cross-matched blood. In such instances, the hematocrit is not an accurate indication of the volume of the intravascular fluid, and other methods of estimation of loss must be utilized. The acute loss of 15 per cent to 30 per cent of the blood volume produces compensated shock, and a 30 per cent to 50 per cent loss results in decompensated shock. Therefore, a patient with symptoms typical of compensated shock should be given approximately one-third of his estimated normal blood volume and should receive one-half of the normal blood volume for compensated shock. When severe acute hypotension has been present for even a very few hours, it may be necessary to replace amounts equal to the entire blood volume. The dangers of incomplete replacement are far more hazardous than those of plethora. With the use of multiple transfusions of citrated blood, it is advisable to add 1 gm. of calcium gluconate for each 1000 ml. of blood used to counteract the citrate effect.

ACUTE RENAL FAILURE

Inadequately corrected hypovolemic vasoconstriction intensified by the action of bacterial toxins and end products[12] results in acute renal failure. This is rarely encountered with extrarenal infections in the absence of renal ischemia. Such tubular damage also occurs as a result of excretion of blood and muscle pigments in the presence of an oliguria.[3]

When initially calculated replacement therapy has been given and the renal response does not rise well above 20 ml. per hour, a renal loading or infusion test should be performed. An increase in the hourly urine output indicates that replacement has been incomplete and that renal function is present. This test is accomplished by the rapid administration of a replacement fluid. The type of fluid used in the infusion test, as in repletion therapy, is based on the clinical analysis of the type of fluid lost. For hemorrhage, 500 ml. of whole blood is used. For losses of water, salt and protein, 500 ml. of plasma or 1000 ml. M/6 sodium lactate is used. In each instance the infusion should take approximately 30 to 45 minutes and the effect on the urine output noted. Venous pressure should be carefully monitored during this period. Quantitative determinations of urea and sodium concentrations in the urine may aid in establishing the presence of acute renal failure. In an oliguric patient, a pronounced reduction in urine urea and high concentrations of sodium are suggestive of impaired tubular function.[3]

In cases in which acute renal failure is the cause of oliguria, the basic objective in management[5,10] is the maintenance of body fluid volume and com-

position at normal levels until the tubular lesion heals and normal renal function returns. Upon diagnosis of this lesion, water intake is immediately restricted. Initially 400 ml. of water plus volumetric replacement of externally lost fluids are administered during the first 24 hours. Water should be given in amounts that will result in a weight loss of a half pound to one pound daily and maintenance of serum sodium concentration at normal levels. If the serum sodium is low, additional water restriction is indicated. Electrolyte losses from the intestinal tract are replaced by physiological solutions of saline.

High caloric intake must be maintained. A patient receiving parenteral fluids on a restricted water regimen may be given 400 ml. of concentrated dextrose daily. The increased dextrose reduces protein catabolism. Drugs excreted by the kidneys, such as streptomycin and digitalis, should be discontinued or limited to avoid toxic levels.

Peritoneal dialysis or extracorporeal hemodialysis is essential for the treatment of symptoms of uremia or potassium intoxication. Recently, the tendency is to institute these measures in four to six days for the prevention of these two situations.

With tubular healing and diuresis, it is essential to measure the daily sodium and potassium losses in the urine. Restoration of the losses guided by this knowledge will prevent serious deficits. The amount of water restored should be somewhat less than the calculated losses in order to avoid exogenous maintenance of the diuresis.

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